Review Article

Peri-implantitis: effects of periodontitis and its risk factors—a narrative review

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Background and Objective: Replacement of missing teeth using implant-supported restorations is a predictable therapeutic modality with reported dental implant success rates of greater than 90–95%: however, implant failures and peri-implantitis still occur. There is a myriad of causes for immediate, early, and late dental implant failures, including the development of peri-implant diseases. This manuscript aimed to describe and discuss the causative factors and risk factors associated with dental implant failures and peri-implantitis, with an emphasis on the relationship of periodontitis and peri-implantitis.

Methods: Narrative overview of the current evidence on risk factors and contributing factors of peri-implant disease, and the association between periodontitis and peri-implant disease retrieved from searches of MEDLINE, PubMed, and Cochrane databases from 1983 to 2021. Articles selected include narrative reviews, meta-analyses, and clinical trials published in English.

Key Content and Findings: The current evidence obtained from through literature search incadetes that the incidence of peri-implantitis is increasing and its severity has similar risk factors to periodontitis including, but not limited to, adverse changes within the oral biofilm, uncontrolled Type-2 diabetes, and an unexplained genetic predisposition.

Conclusions: Peri-implant diseases have a complex etiology and pathogenesis which parallels periodontitis. Both have mutual risk factors/indicators including a dysbiosis of the biofilm, poor compliance with maintenance, enhanced inflammatory responses, smoking and diabetes. However, peri-implant diseases have other unique risk factors, including role of residual cement, peri-implant hard and/or soft tissue deficiencies, prosthetic designs, and potential for novel microbial pathogens.

Keywords: Peri-implantitis; risk factors; peri-implant mucositis

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Introduction

Replacement of missing teeth using implant-supported restorations is a predictable therapeutic modality (1-3). Implant survival rates of greater than 90–95% have been reported for a variety, of patient populations and treatment scenarios (1-4). Although osseointegration can be predictably achieved (5,6), the survival of dental implants is no longer considered a success (7). Instead, the success of implant therapy depends on several elements that influence the implant-prosthetic complex including health and stability of peri-implant soft and hard tissues (7). Peri-implant

Table 1 The search strategy summary

Items	Specification
Date of search	12/1/2020 to 05/20/2021
Databases and other sources searched	MEDLINE, PubMed, and Cochrane databases
Search terms used	"Peri-implantitis", "Periodontitis", "Etiology" and "Risk factors"
Timeframe	From the year 1983 to the date of search
Inclusion and exclusion criteria	Narrative reviews, meta-analyses, or clinical trials published in English
Selection process	The selection process was done independently by two authors and disagreements regarding the selection, if any, were resolved through discussion and consensus

diseases, such as peri-implant mucositis and peri-implantitis, are plaque-associated inflammatory lesions in the tissue surrounding a dental implant that affect the stability of peri-implant soft and hard tissues. We present the following article in accordance with the Narrative Review reporting checklist (available at https://fomm.amegroups.com/article/view/10.21037/fomm-21-63/rc).

Methods

Articles were selected and reviewed from the MEDLINE, PubMed, and Cochrane databases, with keywords such as "peri-implantitis", "periodontitis", "etiology" and "risk factors" as search criteria in order to identify relevant manuscripts. Articles selected include narrative reviews, meta-analyses, and clinical trials. Full text reports published in English from the year 1983 to 2021 were selected. *Table 1* presents more detailed searching process.

Definitions

Peri-implant mucositis is considered as analogous to gingivitis (8). According to the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions, peri-implant mucositis is defined as "*a disease that includes inflammation of the soft tissues surrounding a dental implant, without additional bone loss after the initial bone remodeling that may occur during healing following the surgical placement of the implant*" (9). It is a reversible inflammatory condition that is confined to the soft tissues surrounding an implant without affecting the supporting bone (8,10). The main characteristics of peri-implant mucositis are bleeding on gentle probing and absence of radiographic bone loss following the initial bone remodeling (10). In addition, erythema, swelling and/or suppuration may be clinically evident (8,11).

Peri-implantitis, on the other hand, is analogous to periodontitis. It is considered as the main cause of implant failure after osseointegration (12). Peri-implantitis is defined as "*a plaque-associated pathological condition occurring in tissues around dental implants, characterized by inflammation in the peri-implant mucosa and subsequent progressive loss of supporting bone*" (9). It is an irreversible condition that is diagnosed by presence of clinical signs of inflammation, bleeding on probing and/or suppuration, increased probing depths and/or recession of peri-implant mucosa as well as by presence of radiographic bone loss compared to previous examinations (13). In clinical situations where the clinical or radiographic data from the previous examinations are not available, the diagnosis of peri-implantitis can be made by presence of bleeding on probing and/or suppuration, pocket depth of 6 mm or greater, and peri-implant crestal bone loss of 3 mm or greater from implant shoulder (13,14).

Peri-implant mucositis and peri-implantitis are very common and their prevalence is increasing (15-17). The reported prevalence of these implant diseases has varied among studies since there has not been a uniform diagnostic threshold across studies. A meta-analysis published by Lee and colleagues reported that implant-level and patient-level peri-implantitis prevalence was 9.25% and 19.83%, respectively. Furthermore, they found that implant-level and patient-level peri-implant mucositis prevalence was 29.48% and 46.83%, respectively (15). Another meta-analysis published by Derks and colleagues reported that the implant-level prevalence of peri-implant mucositis and peri-implantitis was 43% and 22%, respectively (16). A systematic review published by Atieh and colleagues also reported that 64% of the patients and 31% of implants were affected by peri-implant mucositis. Moreover, it was reported that 18.8% of the patients and 9.6% of implants

were diagnosed with peri-implantitis (17). The prevalence of peri-implantitis also has been assessed in various patient populations. Monje and colleagues reported that the risk of developing peri-implants was 86% less in patients who attended at least two maintenance visits per year compared to non-compliant patients (18). Dreyer and colleagues reported that the prevalence of peri-implantitis at implant-level was 9.0% for patients who regularly attended maintenance visits and 18.8% for patients without regular preventive maintenance visits. Besides, they reported that the prevalence of peri-implantitis at implant-level was 14.3% for subjects with a history of periodontitis (19).

It is estimated more than 5 million dental implants were placed each year in the United States alone (http://www.ada.org/~/media/ADA/Publications/Files/ADA_PatientSmart_Implants.ashx). Therefore, more than a million implants can be affected by peri-implantitis each year. Hence, it is crucial to detect early signs of peri-implant diseases and to identify patients at a higher risk for developing these diseases.

Etiology and risk factors/indicators

The current evidence suggests that peri-implantitis is preceded by peri-implant mucositis (9). The etiology of peri-implant mucositis is bacterial plaque accumulation around the dental implant (10). However, the histopathologic and clinical factors resulting in the progression of peri-implant mucositis to peri-implantitis are not yet fully understood. Several risk factors or risk indicators have been associated with an increased risk of developing peri-implantitis including the history of periodontitis, poor plaque control, lack or irregular maintenance care, and smoking (13). Interestingly, all these factors are also known risk factor/indicators for periodontitis (20). Hence, in the present review, we further discuss the link between peri-implantitis and periodontitis.

The link between periodontitis and peri-implantitis

The association between periodontitis and peri-implantitis is well studied in several longitudinal and observational studies (21-29). Karoussis and colleagues compared the outcomes of implant therapy in patients with or without history of periodontitis in a 10-year longitudinal study. They demonstrated that the incidence of peri-implantitis at implant-level was 28.6% in patients with history of periodontitis, while it was 5.8% in patients without history of periodontitis (21). Another longitudinal study published by Roccuzzo and colleagues evaluated the outcomes of implant therapy in periodontally healthy patients and in moderately or severely periodontally compromised patients over a 10-year follow-up period. It was found that the incidence of peri-implantitis was significantly different between the groups. It was only 1.7% for patients without history of periodontitis, while it was 15.9% for moderately periodontally compromised patients and 27.2% for severely periodontally compromised patients (22). Several cross-sectional studies have also reported similar outcomes showing greater risk of developing peri-implantitis in patients with histroy of periodontitis with odd ratios ranging between 2.2–9.2 (24-29).

How close is the link between peri-implantitis and periodontitis with regard to effects of the oral microbiome, factors related to lifestyle, and genetic features?

Oral microbiome

Numerous studies have assessed the microbial profile of sites with peri-implantitis using conventional DNA probes or next-generation sequencing technologies such as 16S rRNA-based microarray method (30-39). Presence of periodontal pathogens at sites with peri-implantitis has been reported in several studies, and similarities in microbial profile between periodontitis sites and peri-implantitis sites are well documented (33-38,40). Similar to periodontitis, an increased level of gram negative anaerobic species, and specifically greater levels of *T. forsythia* and *P. gingivalis*, are found at sites with peri-implantitis compared to the sites with implant health (13,32). However, it has been shown that mere presence of p. pathogens at p. sites does not always equate with per-implant bone loss (40).

Next-generation sequencing technologies have enabled researchers to further assess the diversity of the microbiota associated with peri-implantitis by detecting non-cultivable organisms. More recent studies that used 16S rRNA-based microarray method have provided data suggesting peri-implant sites are distinct ecological niches (39). These studies suggest that there are some differences in microbiota between peri-implantitis and periodontitis sites. Particularly, implant surface can act as a modifier of peri-implantitis niche, and lower diversities in microbiota is noted in sites with peri-implantitis compared to those with periodontitis (39). Although there are some distinctions in microbiota between sites with peri-implantitis and periodontitis, overall, these studies have shown similarities in the virulence

characteristics of microbial communities of peri-implantitis and periodontitis. Therefore, these similarities in microbial communities of peri-implantitis and periodontitis may explain the higher incidence of peri-implantitis in patients with the history of periodontitis.

Lifestyle-related factors

There are several lifestyle-related factors that are recognized as risk factors/indicators for both peri-implantitis and periodontitis such as poor plaque control, lack or irregular supportive periodontal therapy, and smoking (13).

It is crystal clear that poor oral hygiene is a risk factor for developing periodontitis (20). In addition, it is well documented that patients with poor oral hygiene are at greater risk for developing peri-implantitis with an odds ratio as high as 14.3 (41). Lack or irregular supportive periodontal therapy has been also shown to increase the chance of recurrence of periodontitis and tooth loss (42,43). The explanation for this finding is that periodontal pathogens can repopulate periodontal pockets within weeks after the active periodontal therapy (44). Several studies have reported similar findings for peri-implantitis (22,23,45,46). Costa and colleagues compared the incidence of peri-implantitis in patients with or without supportive periodontal therapy in a longitudinal study with a 5-year follow-up period. All patients presented with peri-implant mucositis at the baseline. After five years, the incidence of peri-implantitis was significantly different between the two groups, and it was 18.0% is subjects with supportive periodontal therapy and 43.9% in patients without supportive periodontal therapy. Interestingly, the results of this study also demonstrated that presence of periodontitis was associated with significantly greater chance of developing peri-implantitis in both groups with an add ratio of 9.2 (23). Thus, it is extremely important for clinicians to ensure that patients receiving dental implants, especially those with history of periodontal disease, undergo regular periodontal supportive therapy. Furthermore, patients must be educated on effective plaque control techniques, and their ability to clean the implant site should be considered when planning the implant positioning and prosthesis design.

Smoking is another lifestyle-related factor that is considered as a risk factor/indicator for both periodontitis and peri-implantitis (20,21,45,47-50). Smoking affects periodontium directly and indirectly by impairing various neutrophil functions, affecting cytokine production, impairing humoral immune response, inducing microvascular vasoconstriction and fibrosis, and increasing the level of periodontal pathogens in periodontal pockets (20). These changes not only can increase the susceptibility of subject to periodontitis, but also can increase the risk of developing peri-implantitis. A greater risk of developing peri-implantitis has been reported in smoker compared to non-smokers with the odds ratio ranging from 3.6 to 4.6 (49,50). Moreover, a study published by Rinke and colleagues reported a patient-level prevalence of 11.2% for peri-implantitis in a private practice setting. Interesting, they reported that in a small sub-group of patients who were smokers and non-complaint with maintenance therapy, six out of seven patients (85%) developed peri-implantitis (45). However, the effect of the frequency of smoking of risk of developing peri-implantitis needs to be further studied.

It is important to identify lifestyle-related factors in patients with history of periodontitis since same factors can increase the risk of developing peri-implantitis. In addition, clinicians should inform the patients regarding these lifestyle-related factors and their possible effect on the outcome of implant therapy.

Genetic features

It is well documented that periodontal disease is affected by genetic factors. Polymorphism in several genes such as interleukin-1, interleukin-6, interleukin-10, vitamin D receptor, and CD-14 genes have been shown to be associated with periodontitis susceptibility (20). Among these genes, Interleukin-1 polymorphisms and its effect of susceptibility to periodontitis is most widely studied. A meta-analysis published by Karimbux and colleagues demonstrated that Interleukin-1A and Interleukin-1B genetic variations are significantly associated with the increased risk of developing periodontitis in Caucasians with odd ratios of 1.48 and 1.54, respectively (51). In addition, a genome-wide association study has identified 13 genomic noncoding regions (loci) that are associated with increased sub-gingival colonization of periodontal pathogens (52). Recently, it has been reported that there is pleiotropy between periodontitis and cardiovascular diseases, and at least four loci are common between coronary artery disease and periodontitis (53), suggesting these loci may result in aberrant inflammatory pathways and increased susceptibility of an individual to these diseases.

There is still limited evidence available regarding the effect of genetic factors on susceptibility to peri-implantitis. A cross-sectional study published by Laine and colleagues

reported an association between interleukin-1 receptor antagonist (IL-1RA) polymorphisms and prevalence of peri-implantitis with an odd ratio of 3 (54). Similar findings was reported for a positive IL-1 composite gene polymorphism (IL-1α –889; IL-1β +3954) in a cross sectional study of only 50 patients (55). However, further studies especially on a genome-wide level are needed before a definitive conclusion can be drawn. It is recommended that patient with a history of periodontitis be informed about higher chance of developing peri-implantitis as they may be genetically more susceptible to other inflammatory conditions such as peri-implantitis.

Localized predisposing factors

In the case of plaque-associated peri-implantitis, prosthetic and site specific anatomical factors have been shown to be predisposing factors in biofilm adherence around dental implants, thus leading to inflammation and peri-implantitis (56).

Prosthetic factors such as the presence of residual cement, prosthetic connections emergence profile of the restoration, and implant positioning have all been shown to be a contributing factor in the prevalence of peri-implant diseases (56). Through systematic reviews, excess or retained cement has been identified as a possible risk indicator for the development of peri-implant diseases (57). The presence of residual cement favors bacterial attachment and inflammation due to the increased surface roughness, which leads to a higher incidence of peri-implant diseases. Increased incidence of residual cement has been associated with over contoured restorations, restorations with concave surfaces and restorations at a sub-mucosal margin (56,57).

Factors within the prosthetic restoration, including the implant-abutment connection and the emergence profile of the restoration, can contribute to the development of peri-implantitis (56). Implant abutment connections can vary depending on implant system and can be classified as a no interface, platform switched, conical connection and butt-joint connection. The presence of a micro-gap within the implant abutment interface, allows for bacterial colonization, leading to gingival inflammation and peri-implant bone loss (58). Higher incidence of peri-implant bone loss (1.5–2.0 mm) has been seen with butt-joint implant-abutment connection as compared to a platform switched interface due to the reduction in the size of the micro-gap (58). Emergence profile of crown can impact the peri-implant tissues; over contoured restorations and restorations with a concave emergence profile can lead to an increased risk of bone loss over time due to the increased bacterial adherence and decreased cleansibility (56,59).

In addition to prosthetic related factors, implant placement and positioning and the role of hard and/or soft tissue deficiencies is another important predisposing factor in the etiology of peri-implant diseases. Hard and/or soft tissue deficiencies are a common occurrence at implant sites, and if not properly identified and corrected can lead to increased marginal bone loss, soft tissue inflammation and/or soft tissue recession over time (60). Hard and/or soft tissue deficiencies can be present either before implant placement (e.g., Resorption due to tooth loss, infection, periodontitis, trauma, vertical root fracture, etc.) or can occur after implant placement (e.g., malposition of implant placement, systemic disease, peri-implantitis, lack of buccal bone etc.) (60).

Documented inter-relationships (meta analyses)

Dental implants have a long history of documented success, with 3 and 5-year success rates of 99.12% and 97.38%, respectively, and an even higher survival rate of 99.26% after 5 years in a sample size of 990 implants placed in 590 patients (61). Dental implant success criteria have been described by Albrektsson *et al.* as lack of mobility of the implant, no radiographic evidence of peri-implant radiolucency, less than 0.2 mm bone alveolar bone 1-year post implant placement, and absence of persistent pain or infection (62). Many newer published dental implant success criteria do not include the annual bone loss as a criterion as newer implant designs have largely reduced or eliminated successive bone loss after loading (63,64).

Updates in the success criteria of dental implantology are currently separated into 4 distinct levels including success at the implant level, peri-implant soft tissue level, the prosthetic level and the patient satisfaction level (7). For the purpose of this review, the focus will be on the implant level in which the success criteria largely remain unchanged since 1986 when first reported by Albrektsson *et al.* except for the successive bone loss that occurred with earlier implant designs and may no longer be a factor. Newer studies demonstrate that conical interface implants and platform switching greatly reduce early marginal bone loss (64).

Despite the high success rate that is documented in the literature, dental implant failures still occur and can be separated into two distinct categories: early and late failures. Early failures occur prior to the establishment of

osseointegration or the inability to achieve it. Late failures occur after achieving osseointegration and are most often due to severe peri-implantitis (65). Etiology of early failures can be categorized into iatrogenic and patient related factors. Iatrogenic factors leading to early failure include overheating of the osteotomy during placement, placement into a vital structure, possible surgical site contamination causing an infection, improper proximity to adjacent teeth or implants, improper implant angulation causing thinning or loss of buccal/facial bone, and lack of primary stability among others. During implant placement, overheating the osteotomy to a temperature of 47 degrees or high for more than a minute has been associated with necrosis, leading to failure to osseointegrate (66). Placement of an implant into vital structures that may cause paresthesia, pain or infection would also lead to an early or immediate failure that will necessitate implant removal (67). Other factors for early failure include the lack of primary stability during dental implant placement (68). Manzano *et al.* performed a meta-analysis of 18,171 implants and found that implants shorter than 10 mm is a risk factor for early failure along with smoking and implants placed in the maxilla (68). However, more recent studies have shown that success rates for shorter implants have gradually increased from the 1990s to 2010s. Patient related factors include a history of uncontrolled diabetes, chronic periodontitis, smoking, location of the implant and bone quality and quantity (69,70). Incidence of early failures occurred in 1.4% of the dental implants placed in a longitudinal study of 596 patients with 2,765 implants (3). In a meta-analysis of 73 studies, early implant losses occurred in 3.60% of 16,935 implants with surgical trauma and anatomical conditions as the most often cited factors for failure (71).

Late implant failures are defined as loss of the implant after achieving osseointegration (65). Late failures are predominantly due to the presence of biological factors leading to alveolar bone loss and the clinical presentation of peri-implantitis. Other less common causes of late implant failures include implant fracture, which has been documented to occur less than 0.2% annually (72). Patients with a history of periodontitis have an implant failure by an odds ratio of 3.02 compared to periodontally healthy patients (73). There are suggestions in the earlier literature stating that shorter dental implants (<7 mm) may have an increased failure rate (72), however, there are much more overwhelming data suggests that shorter implants have the same success rate as standard length implants (74-76).

One meta-analysis of 16 studies found that narrow diameter implants (<3.3 mm) had a significantly lower survival rate of 75% compared to implants with diameters greater than 3.3 mm, which had a survival rate of 87% (77).

The survival of dental implants placed in the maxilla appears to be more than 3 times less than those placed in the mandible in fully edentulous patients (71). The location of dental implant placement in the dental arch has been investigated as a possible predictor for implant success. In 1992, Drago *et al.* published a study on rates of osseointegration based on anatomic location of implant placement of 673 fixtures. He found that the highest implant osseointegration was in the posterior mandible (98.7%), followed by the anterior mandible (96.7%), anterior maxillae (89.1%) and posterior maxillae (71.4%) (78). This coincides with a higher incidence for surgical intervention for peri-implantitis for maxillary implants when compared to implants placed in the mandible (79).

Several outcome studies have focused on the effect of the implant restoration on the survival and success of the implant. In a cross sectional study, Dalago *et al.* found that patients rehabilitated with full arch implant restorations had an increase odds ratio of 16.1 of developing peri-implantitis when compared to single fixed dental prosthetic restorations (29). The impact of different restorations on the success rate of dental implants was investigated in a prospective clinical trial of 630 patients and 1,569 implants. In his study, implants restored with single crowns had the highest success after 5 years (97%), followed by fixed dental prostheses (95.5%) and removable dental prostheses (93%) (80).

Since the inception of dental implants by Professor Branemark in 1965, the replacement of missing teeth with dental implants has gained huge popularity and the number of dental implants placed per year has dramatically increased. In 1988, it was estimated that approximately 100,000–300,000 dental implants were placed per year in the United States (81). However, based on the latest market research, an estimated 1,260,000 dental implant procedures were performed in 2013 in the United States, and this number is projected to double in 2020 (82). With the increasing number of implants placed by clinicians, the raw number of incidences of implant failures and peri-implant diseases is expected to increase (83). Based on implant therapy outcome studies that were published before and after the year 2000, there has been an increase in the 5-year survival rate of dental implants from 93.5% to 97.1%. Despite this increase in survival rate, the incidence of peri-implantitis has not changed significantly (5-year biologic

complication rate of 3.3% in older studies compared to 2.5% in newer studies) (84). However, due to the increase number of implants being placed, the absolute number of implants with peri-implantitis has increased.

Conclusions

Based on the current body of evidence, peri-implant diseases, peri-implant mucositis and peri-implantitis, are diseases affecting dental implants with a complex etiology and pathogenesis with similarities to that seen in periodontitis. Periodontitis and peri-implantitis have mutual risk factors and risk indicators including poor plaque control, lack or irregular maintenance therapy, smoking and diabetes. Peri-implant diseases have other unique risk indicators and contributing factors, not seen in periodontitits, which contribute to the complex nature of the etiology of these diseases.

Local predisposing factors are often responsible for site-specific diseases. Local contributors, such as surgical and prosthetic variables, together with soft and hard tissue characteristics, may be predisposing factors in the event of plaque-associated peri-implantitis, which results in inflammation.

Recent advancements in metagenomics may make it possible to better identify the specific pathogens responsible for peri-implant disease, which could pave the way for new therapeutic strategies. Recent microbiological discoveries have shed fresh light on the etiology of peri-implant diseases. The development of prospective novel therapeutic methods (such as the creation of a microbiota transplant therapy) to use in the treatment of peri-implant disorders may result from a complete understanding of oral and peri-implant microbiota in health and disease in its full genetic composition. For clinicians to better understand, prevent the occurrence of, and eventually cure peri-implant diseases, this review provides a comprehensive overview on the current body of evidence available on risk factors, risk indicators and local predisposing factors on a surgical, prosthetic and patient level for clinicians to better understand, prevent the occurrence of and ultimately treat peri-implant diseases.

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